Using Topically Applied Diuretics to Bring about Tissue-Specific Dehydration in Conjunction with NSAIDs to Enhance Nerve Adhesion for Pain Treatment at Lower Dosages

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Simon Edwards
Research Acceleration Initiative

Introduction

Seeking to explain substantial variation in efficacy of NSAID pain relievers between individuals and even between usage instances in the same individual, I have identified local tissue hydration as an antagonist of analgesic efficacy and thus speculate that bringing about local dehydration in a tissue at any point after an NSAID is introduced to the blood stream would enhance adhesion to nerve fibers and thus provide superior analgesic effects at the same dosage.

Abstract

If we think of the diffusion of analgesic compounds in the body as having some similarities to algae floating on the surface of a stagnant creek and we think of the nerve fibers as being an object sitting on the creek bed that is only exposed when the water level drops below a certain point, if the goal is to try to coat that object with algae, we might bring that about by temporarily lowering the water level.

Analgesic inefficacy has been alternatively attributed to malabsorption, development of drug tolerance, individual pain sensitivity, and in the case of abscessed teeth and infected tissues, extreme inflammation of tissues preclude the entry of analgesic compounds into the critical area due to its hydrostatic pressure being kept constantly higher. If this is the reason for non-efficacy of analgesics such as ibuprofen in the case of abscess, it stands to reason that the opposite should be true; that lowering local hydration should increase adhesion.

Although general systemic hydration is desirable and helpful in general diffusion of oral-route analgesics of the NSAID class, never before has the induction of local dehydration about one hour post-ingestion been studied for its potentiating effects. This research area is unexplored and deserving of study as a sudden deficit of hydration in a local area brought about once the analgesic compound has diffused throughout the body should generate large numbers of adhesion events in the innervated areas with repeated cycles of diuresis further increasing the effect as would be expected in any process of layered deposition.

This effect may shed light on the improved efficacy of NSAIDs such as ibuprofen and naproxen versus non-NSAIDs such as acetaminophen since the first step toward improving local adhesion where inflammation is present is the reduction of inflammation. If getting analgesics to adhere to inflammed tissues is like trying to putt a golf ball up a hill and hoping it will come to rest at the exact apex of that hill, Local Diuresis-Enhanced Analgesic Adhesion

(LDEAA) is akin to the much easier task of trying to get that same golf ball to roll into a divet. This novel strategy may help to alleviate suffering and reduce health care costs associated with NSAID overuse as well as opioid use.

Conclusion

If successful, pain treated by an LDEAA approach would be the equivalent of taking 10x the dose of pain medication with none of the side-effects and the retention of the ability to feel pain in untreated areas, reducing the risk of sustaining injuries resulting from general analgesia.